

Determination of the acid-base status in 50 horses admitted with colic between December 1998 and May 1999

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Abstract — The purpose of the present study was to investigate the acid-base status and the concentration of organic acids in horses with colic caused by various disorders. Blood samples were collected from 50 horses with colic and from 20 controls. No intravenous fluids had been given prior to sample collection. Identified causes of colic included gastric ulceration, small intestinal volvulus, cecal intussusception, cecal rupture, colonic impaction, left dorsal colon displacement, right dorsal colon displacement, colonic volvulus, colitis, peritonitis, and uterine torsion. Thirty-seven horses recovered from treatment of colic, 8 horses were euthanized, and 5 died. Most cases were not in severe metabolic acidosis. In previous studies, most horses presented for diagnosis and treatment of colic were in metabolic acidosis and in shock.

Résumé — Détermination de l'état acido-basique chez 50 chevaux admis pour colic entre décembre 1998 et mai 1999. L'objectif principal de cette étude était d'évaluer l'état acido-basique et la concentration des acides organiques de chevaux admis pour le traitement de colique entre la période de décembre 1998 et mai 1999. Du sang a été collecté sur 50 chevaux en colique et 20 chevaux en santé. Aucune fluidothérapie intraveineuse avait été administrée à ces chevaux avant la prise de sang. Les coliques furent associées à des ulcères d'estomac, volvulus du petit intestin, intussusception du caecum, rupture de caecum, impaction du colon, déplacement dorsal et vers la gauche du colon, déplacement dorsal et vers la droite du colon, volvulus du colon, colite, péritonite, et torsion utérine. Trente-sept chevaux ont bien répondu au traitement des coliques, mais 8 chevaux ont été euthanasiés et 5 sont morts. Même si les concentrations d'acides organiques furent élevées chez les chevaux en colique, ces chevaux ne présentaient pas pour la plupart une acidose métabolique sévère. Nos résultats diffèrent des études publiées antérieurement où plusieurs chevaux étaient en acidose métabolique et en état de choc à l'admission.

(Traduit par docteur Germain Nappert)

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Introduction

Colic is one of the most frequent problems encountered by equine veterinarians (1,2). There are numerous causes of colic, including intestinal and non-intestinal disorders. A previous study demonstrated metabolic acidosis in 83.3% of horses with acute diarrhea and in 66.7% of horses with serious forms of colic (3). Dehydration is a frequent cause of metabolic acidosis (4,5). Dehydration leads to hypovolemia and low tissue

perfusion, resulting in a limited supply of oxygen to tissues and a decrease in H⁺ excretion by the renal tubules (5). As a further consequence of dehydration, the biosynthesis of the L isomer of lactic acid from anaerobic glycolysis metabolism by skeletal muscle is increased (6,7). The normal plasma L-lactate concentration in horses is generally considered to be < 1.5 mmol/L (8). Increases in plasma L-lactate concentration have been categorized as mild (2.5 to 4.9 mmol/L), moderate (5.0 to 9.9 mmol/L), and severe (> 10 mmol/L), with a low probability of survival in horses affected with colic when the blood lactic acid is > 6.72 mmol/L (9,10). An increased amount of plasma L-lactic acid in 14 horses with intestinal disorders was held responsible, in part, for a high anion gap (11). A high anion gap has also been associated with an unfavorable prognosis in horses affected with colic (12). However, the high anion gap in horses with intestinal disorders has never been completely explained by an increase in whole blood lactate, pyruvate, hydroxybutyrate, or acetooacetate concentrations (11).

An increased concentration of D-lactate, the stereoisomer of L-lactate, in acidic diarrheic calves without

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signs of dehydration has been reported recently (13,14). It is believed that malabsorptive diarrhea could promote bacterial fermentation of undigested nutrients in the ileum and the large intestine, resulting in the production of D-lactate by abnormal gastrointestinal flora and its subsequent absorption into the blood. In adult ruminants, the bacterial fermentation of starch resulting in hyper D-lactic acidosis has been recognized as a consequence of grain overload (6), where substantially more D-lactic acid is produced by *Lactobacillus* organisms than is L-lactic acid by microbial fermentation (15). Overproduction of D-lactic acid by intestinal bacteria could also occur in the colon, in a manner similar to human D-lactic encephalopathy, which occurs as a complication of the short bowel syndrome (16). The accumulation of D-lactate has also been observed in cats with diabetes mellitus (17). Since mammalian cells do not possess the enzyme D-lactate dehydrogenase, which is responsible for the production of the D-lactate (18), only limited amounts of D-lactate can be produced by mammalian cells through the glyoxalase pathway (19). Some D-lactate, after transformation into pyruvate, is oxidized or utilized for gluconeogenesis. D-Lactate is usually excreted in the urine, but renal clearance is much slower for D-lactate than for L-lactate (20).

The purpose of the present study was to investigate the acid-base status and concentration of organic acids in horses admitted between December 1998 and May 1999 for treatment of colic caused by various disorders. We hypothesized that most horses admitted for treatment of colic at our clinic are in metabolic acidosis. Measurement of organic acids by high performance liquid chromatography may allow us to better explain metabolic imbalances and increased anion gap in critically ill horses, which could have considerable impact on our thinking, not only about the causes of acidosis, but also about its management.

Materials and methods

Horses admitted to the Veterinary Medical Teaching Hospital, University of Missouri-Columbia, for treatment of colic caused by various disorders were used in the study. Signs of colic in the population studied included restlessness, lying down and getting up, rolling, and kicking at the abdomen. At admission, 3 mL of blood were collected anaerobically from the jugular vein in a preheparinized plastic syringe (Smooth-E; Radiometer America, Westlake, Ohio, USA). For the determination of organic acid concentrations, a second 3-mL blood sample was collected and placed in a tube without anticoagulant (Vacutainer; Becton-Dickinson, Rutherford, New Jersey, USA). On clotting, serum was separated by centrifugation and frozen at -20°C until analyzed. Control blood samples were similarly collected from 20 healthy horses.

Acid-base parameters (pH; bicarbonate (HCO_3^-), potassium (K^+), and sodium (Na^+) concentrations; total CO_2 ; partial pressure of CO_2 ; and base excess) were determined within 15 min of blood collection by using an automated blood gas analyzer (Sendx 100; Sendx Medical, Carlsbad, California, USA). The blood gas analyzer was calibrated every 24 h with buffer solutions that were provided by the manufacturer. The blood

pH measurements with the blood gas analyzer were corrected for the rectal body temperature. Serum albumin, chloride, and creatinine concentrations were determined simultaneously with an automatic chemistry analyzer (Vitros 500; Johnson & Johnson Clinical Diagnostic, Rochester, New York, USA). Anion gap was calculated as the difference between the sum of the serum concentrations of the measured cations ($\text{Na}^+ + \text{K}^+$) and the measured anions ($\text{Cl}^- + \text{HCO}_3^-$). Serum acetate, pyruvate, and β -hydroxybutyrate concentrations were determined by using the ion exclusion method for the separation of organic acids (21). The nonstereospecific assay has also been found to be suitable for measuring the racemate DL-lactate, a compound containing both enantiomers of lactic acid (22). The stereospecific analysis of serum lactate enantiomer concentrations was accomplished by high performance liquid chromatography (HPLC) (22). The HPLC system consisted of a pump (Waters Model 626; Millipore, Milford, Massachusetts, USA), a tunable wavelength UV absorbance detector (Waters 2487; Millipore) and an autoinjector (Waters 717 plus; Millipore). Data collection, integration, and calibration were performed using a chromatography manager (Waters Millennium Version 32; Waters, Millipore).

Statistical analysis

Data were analyzed with a statistical computer program (Systat v. 8.0; SPSS, Chicago, Illinois, USA). The mean concentration of all organic acids was calculated for the control horses and those with colic. An unpaired, 2-sample *t*-test was used to compare blood gas components and measured concentrations of lactic acid, enantiomers, and other organic acids. The separate variance test value was used when unequal variances were found for some of the parameters. In addition, analysis of variance was used for comparisons of organic acid concentrations in the serum for the controls, horses with colic that recovered, and horses with colic that were euthanized or died. All values were expressed as the mean and standard deviation (*s*); a *P* value of less than 0.05 was considered significant.

Results

Age, gender, and physical assessment

Average age of horses was 11, *s* = 7 y, and 10, *s* = 8 y, in controls and horses with colic, respectively. Two stallions, 7 geldings, and 11 females were included in the control group. Six stallions, 20 geldings, and 24 females were included in the group with colic. Identified causes of colic included gastric ulceration (*n* = 3), small intestinal volvulus (*n* = 9), cecal intussusception (*n* = 1), cecal rupture (*n* = 1), colonic impaction (*n* = 17), left dorsal colon displacement (*n* = 3), right dorsal colon displacement (*n* = 1), colonic volvulus (*n* = 5), colitis (*n* = 6), peritonitis (*n* = 3), and uterine torsion (*n* = 1). Thirty-seven horses recovered from treatment, but 8 horses were euthanized, and 5 died.

Blood gas, serum concentration of organic acids

Blood gas analysis revealed significant differences in the mean concentrations for serum total CO_2 (TCO_2),

Table 1. Comparisons of blood gas, electrolytes, and serum organic acid concentrations in controls and horses with colic

Blood parameter	Controls	Recovered	Euthanasia/died	Colic
n	20	37	13	50
pH	7.46, s = 0.04	7.44, s = 0.06	7.43, s = 0.12	7.44, s = 0.08
pCO ₂ , mmol/L	42.46, s = 5.72	38.1, s = 8.44	42.37, s = 16.8	39.76, s = 10.61
TCO ₂ , mmol/L	31.06, s = 3.64	27.98, s = 5.2	28.83, s = 5.81	28.24, s = 5.35 ^c
HCO ₃ ⁻ , mmol/L	30.09, s = 3.31 ^a	26.79, s = 5.08 ^{a,b}	25.52, s = 8.11 ^b	26.39, s = 6.12 ^d
Base excess, mmol/L	5.21, s = 2.73	2.75, s = 5	2.44, s = 6	2.65, s = 5.23 ^c
Na ⁺ , mmol/L	134.19, s = 5.34	133.56, s = 3.58	131.51, s = 6.11	132.95, s = 4.52
K ⁺ , mmol/L	3.49, s = 0.58 ^a	3.1, s = 0.45 ^b	3.11, s = 0.76 ^{a,b}	3.1, s = 0.55 ^c
Cl ⁻ , mmol/L	99.05, s = 4.75	98.53, s = 4.99	99.13, s = 9.8	98.71, s = 6.66
Albumin, g/dL	2.5, s = 0.34 ^{a,b}	2.73, s = 0.45 ^a	2.37, s = 0.67 ^b	2.62, s = 0.54
Anion gap, mmol/L	8.45, s = 7.06	10.74, s = 7.23	9.96, s = 16.26	10.49, s = 10.77
Pyruvic acid, mmol/L	0.04, s = 0.04 ^a	0.09, s = 0.08 ^{a,b}	0.14, s = 0.16 ^b	0.1, s = 0.11 ^d
DL-Lactic acid, mmol/L	1.56, s = 2.41 ^a	2.91, s = 2.75 ^{a,b}	4.35, s = 5.52 ^b	3.38, s = 3.8 ^c
L-Lactic acid, mmol/L	0.88, s = 0.86 ^a	2.91, s = 2.85 ^{a,b}	4.02, s = 4.76 ^b	3.29, s = 3.53 ^d
D-Lactic acid, mmol/L	0.71, s = 0.26	0.42, s = 0.32	0.33, s = 0.31	0.8, s = 0.64
Acetic acid, mmol/L	0.16, s = 0.13	0.37, s = 0.97	0.14, s = 0.29	0.3, s = 0.84

Na⁺ — sodium ion, K⁺ — potassium ion, Cl⁻ — chloride ion; anion gap — (Na⁺ + K⁺) - (HCO₃⁻ + Cl⁻); pCO₂ — partial pressure of carbon dioxide

^{a,b}Within a row, means with different superscript letters are significantly (*P* < 0.05) different.

^cSignificantly different blood parameters between controls and horses with colic, *P* ≤ 0.05 (Student's *t*-test)

^d*P* ≤ 0.01

Values are mean and standard deviation (s)

Table 2. Comparisons between the serum L-lactate concentration and the cause of colic in 50 horses

Serum L-lactate concentration	Cause of colic (n)	Outcome
< 2.5 mmol/L	Small intestinal volvulus (1) Colonic impaction (12) Colonic volvulus (2) Left dorsal colon displacement (2) Right dorsal colon displacement (1) Gastric ulceration (3) Peritonitis (2) Acute diarrhea (1)	Euthanasia Survived (11), euthanasia (1) Survived (1), died (1) Survived Survived Survived Survived (1), euthanasia (1) Euthanasia
2.5 to 4.9 mmol/L	Acute diarrhea (3) Small intestinal volvulus (7) Colonic impaction (4) Colonic volvulus (2) Cecal intussusception (1) Cecal rupture (1) Uterine torsion (1) Peritonitis (1)	Survived Survived (4), died (1), euthanasia (2) Survived Died Survived Euthanasia Died Euthanasia
5.0 to 9.9 mmol/L	Colonic impaction (1) Acute diarrhea (1) Left dorsal colon displacement (1)	Survived Survived Survived
≥ 10 mmol/L	Small intestinal volvulus (1) Acute diarrhea (1) Colonic volvulus (1)	Died Survived Euthanasia

bicarbonate, base excess, and potassium between controls and horses with colic (Table 1). The anion gap, however, was not significantly different between the groups. Blood pH did not differ between groups in our study. Horses with colic had higher concentrations of DL- and L-lactic acid, pyruvic acid, and β-hydroxybutyric acid than did controls. Overall, there was a tendency for a greater increase in L-lactate than in D-lactate in horses with colic. Since a high level of β-hydroxybutyric acid was identified by HPLC in several horses with colic, confirmation of this compound was performed by triple quadrupole mass spectrometry (Micromass Quattro IIE; Micromass Ltd, Altrincham,

United Kingdom). Co-eluting peaks were found with glycerin, a humectant of the centrifugal filter unit used to remove protein from samples. Therefore, the HPLC method was inaccurate to measure β-hydroxybutyrate. In comparison with the controls, horses with colic that did not recover from medical or surgical treatment had higher levels of DL- and L-lactic acid, and β-hydroxybutyric acid. The resulting outcome of all the horses with colic is tabulated for 4 ranges of blood L-lactic acid concentration in Table 2. Overall, increases in serum L-lactate concentration were categorized as mild in 20 cases, moderate in 3 cases, and severe in 3 cases.

Discussion

This study represents the first attempt to document the possibility of most horses admitted at our clinic for treatment of colic having metabolic acidosis. In addition, HPLC afforded the opportunity for simultaneous investigation of several organic acids that may contribute to metabolic imbalances.

Venous blood pH is usually considered to be normal between 7.32 and 7.44 (23,24). In this study, the automated blood gas analyzer gave a blood pH of 7.46 for the controls. Normal values for blood gas analysis in horses with this blood gas analyzer have never been established. According to the manufacturer, the machine was designed to measure acid-base parameters accurately, only for human blood (the amounts of HCO_3^- calculated by the automated blood gas analyzer were based on a human formula). Although the machine was calibrated regularly, according to the manufacturer's recommendations, it constantly appeared to overestimate the blood pH and the amounts of HCO_3^- in the equine samples. Small, but significant, decreases in HCO_3^- , TCO_2 , and base excess were found in most horses with colic, suggesting an underlying metabolic acidotic process, although the pH was not affected (23). Hypokalemia in horses with colic was most likely associated with altered intake and absorption or with excessive K^+ losses from the gastrointestinal tract caused by diarrhea (24).

The normal plasma L-lactate concentration is generally considered to be < 1.5 mmol/L in horses (8). Most of the horses with colic in this study presented with a higher level of L-lactate. The L isomer of lactate predominates in mammalian metabolism and is of particular importance in tissue hypoxia (25). In order to meet the continuing needs for adenosine triphosphate during anoxia, anaerobic glycolysis predominates over the aerobic tricarboxylic acid cycle, resulting in the increased production of L-lactate from pyruvate by the action of L-lactate dehydrogenase. The conversion of L-lactate into pyruvate by L-lactate dehydrogenase is possible in all tissues, especially the heart and skeletal muscles. Severe lactic acidosis may also be compounded by decreased hepatic uptake (26) or reduced renal perfusion (25). In addition, increased pyruvate was found in horses with colic in this study. Pyruvic acid concentrations were, however, too low to have a major impact on acidosis.

No major increase of D-lactate was found in our study. In calves, D-lactic acid, which can be produced by bacterial fermentation of milk, is a significant contributor to metabolic acidosis (27). The incidence of D-lactic acidosis in foals with diarrhea has not been studied yet. In addition, lactic acidosis has been shown to occur prior to laminitis in those horses that have been subjected to carbohydrate overload (28,29). These 2 conditions may be worthy of further investigation.

Butyric acid is normally produced by bacterial fermentation in the large colon and modified by the intestinal mucosa into β -hydroxybutyrate before absorption (30). High levels of butyric acid in the blood of horses with colic could represent compromise of the normal barrier between the lumen of the intestine and the blood. The presence of co-eluting peaks with the HPLC method prevented any assumptions for this organic acid being made.

Although some organic acids are significantly increased in horses with colic, most cases presented over a 6-month period at our clinic were not in severe metabolic acidosis. In addition, the production of D-lactate by the large intestine was not a significant concern. Furthermore, some horses with colic were even in metabolic alkalosis at admission. No IV fluids had been given prior to sample collection at the clinic or by referring veterinarians. This finding is not consistent with those of previous studies where most horses presented for diagnosis and treatment of colic were in metabolic acidosis and in shock (3,10,11).

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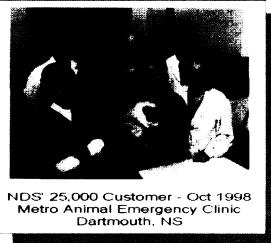
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